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Effects of essential oils on house dust mites

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The efficacy of essential oils and terpenoids in killing human lice has been confirmed by previous in vitro work in our laboratories (Weston et al 1997). This work has now been extended to other arthropods, such as mites, in the hope of discovering a model upon which to screen for pediculicidal activity. It may also reveal that essential oils and terpenoids are toxic to other clinically important species.

Mites cause diseases, such as scabies, and various veterinary infestations. Recently, attention has been diverted to *Dermatophagoides pteronyssinus*, the house dust mite, because of its involvement in asthma. D. pteronyssinus is also easily cultured in the laboratory, making it an ideal choice for the development of a rapid screening procedure for acaricidal compounds. This will be utilised for the bio-assay guided fractionation of plant extracts and essential oils.

Mite colonies were propagated using a method developed at Medical Entomology Centre, Cambridge, (I. Burgess, personal communication) as follows: The sides of the bottom of a 9 cm petri dish were covered with 3 layers of masking tape, so that 1 cm was protruding above the dish, the adhesive exposed inwards. A 3 mm layer of mite colony / substrate (finely ground desiccated liver and brewers' yeast, equal parts by weight) mixture was added to the dish and the lid placed over the protruding tape. The dish was sealed with further tape and incubated at 25 °C and 75 % RH.

In the insecticidal assays employed in our laboratory, filter paper is impregnated with test agent, the insects placed on it and the apparatus set up such that contact between insect and test paper is maintained during the experiment (WHO 1991). Mites are very small, very mobile, and able to climb smooth, dry surfaces. Any object which contacts the filter paper provides an escape route, making continuous contact difficult to ensure. Size and mobility also pose further difficulties of observation and quantification. In the following procedure, a novel method was used which minimised the mites' escape routes from the test papers. This

worked for active acaricides but variable losses due to escape were experienced in the control group. However, since immobilisation would occur prior to mortality, the presence of mites on the papers over time was highly indicative of acaricidal activity.

A 3 cm diameter filter paper was suspended by fine wires in the centre of a short length of plastic tubing. A piece of tubing was prepared for each test agent. The filter papers were then impregnated with either 0.1 ml lavender oil, tea tree oil, or lemon oil, all at 10 % v/v concentrations in absolute ethanol. The control was impregnated with vehicle only. All papers were dried in air for 5 minutes. The lengths of tubing were immediately placed on individual half petri-dishes, in the bottom of which, a layer of tissue paper soaked in 15 ml of saturated saline solution had been placed to provide humidity. Ten dust mites, individually removed from the colony using a fine paintbrush, were placed on each of the test papers. Half petridishes were placed over the tops of the lengths of tubing and the apparatus re-incubated. At 30 minutes, mites were observed (using a binocular microscope at 25 x magnification) for mobility, and at 2 hours for mortality, defined by the lack of movement when touched with forceps. Each experiment was replicated

Tea tree oil was the most effective acaricide, causing 100 % immobility at 30 minutes and 100 % mortality at 2 hours. Lavender oil was acaricidal to a lesser extent giving 87 % immobility at 30 minutes and 87 % mortality at 2 hours. Lemon oil gave 63 % immobility at 30 minutes and 80 % mortality at 2 hours. The control showed 0 % immobility at 30 minutes and 0 % mortality at 2 hours. Full results will be given in the presentation. Further work continues to isolate the most potent constituents of these oils and to determine whether a correlation exists between pediculicidal and acaricidal activity.

Weston, S.E. (1997) J. Pharm. Pharmacol. 49 (suppl 4), 120. WHO/VBC/81.808 5pp World Health Organisation, Geneva 1981.